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ABSTRACT

This paper explores the nature of the aging process by providing an overview of the available evidence relating to the body systems that are most critical to biological function. Each system is treated separately to more clearly describe various aspects of the aging process and then integrated in a discussion of the theories of biological aging. Concepts are presented so they may be understood without a thorough knowledge of biological and chemical terminology. The issue of lifestyle intervention in the aging process is addressed and an examination is made of those components that have been shown to affect physiological aging. (Author/JD)

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Physiological Aging and Exercise

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Although the aging process is a well-accepted biological phenomenon, civilizations since the beginning of time have sought ways to retard this process with the ultimate goal of immortality. It has only been in our relatively recent past that a systematic effort has been made to more clearly understand the scientific implications of the biological process. As life has a beginning, so does it have an end, and as time progresses the cumulative biological changes that occur in all species ultimately result in a decreased ability to function within their environment. The length of time that it takes for the biological changes to occur varies greatly from one individual to another regardless of the absence or presence of complicating pathological conditions. Although the average life expectancy in ancient Greece was approximately twenty-two years, Sophocles wrote some of his best works and achieved great success at the age of eighty-five. Moreover, his death at age ninety clearly illustrates that the maximum life span has not been extended very much since that time. Those who reach a similarly

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advanced age in present society probably experience the same type of biological insufficiency that Sophocles did, even though we have had the advantage of a much greater degree of medical sophistication.

The fact that life expectancy in our present society has reached approximately seventy years of age, simply means that a greater number of our contemporaries live to the eighties and nineties as compared with those of ancient Greece. Better nutrition and more effective treatment of disease has been credited with this added longevity. However, it appears that the key to longevity lies in a more complete understanding of the biological aging process itself. Such understanding would provide us with the knowledge necessary to not only extend life but also to improve the quality of life during advanced years. It is doubtful that individuals of any species actually attained their full potential either in longevity or capacity. Continued study is important so that the aging process can be carefully observed under controlled conditions and thus help a greater proportion of individuals come closer to that potential. The efforts of many of our best biological scientists have provided the bases for several theories attempting to explain the biological aging process. It appears certain that continued research generating a greater amount of supporting information will help us to utilize these theories, which can then be used as a guide to further study and the acquisition of more sophisticated understanding of the aging pro-

cess. Some scientists project that a more thorough knowledge of the aging process may make it possible to extend life to 150 or possibly 200 years. Needless to say, this is a very optimistic prediction but one that appears well within the realm of possibility.

This paper explores the nature of the aging process by providing an overview of the available evidence relating to the body systems that are most critical to biological function. Each system is treated separately to more clearly describe various aspects of the aging process and then integrated in a discussion of the theories of biological aging. It is important to note that, while all of these theories have been developed on the basis of research evidence related to the aging process, some have more documented support than others. The description of both the body systems and theories involve some technical concepts that may be unfamiliar to the non-researcher. However, an attempt has been made to present such concepts so they may be understood without a thorough knowledge of biological and chemical terminology. The final section addresses the issue of lifestyle intervention in the aging process, and includes an examination of those components that have been shown to affect physiological aging.

In most cases, as the aging process continues, a decrease in the intensity and duration of physical activity occurs. That is, physical activity becomes slower and it is less efficient. Rubinstein speculates that these changes

are largely due to disease and nutritional deficiencies.[1] Motor disorders and dysfunctions were first thought to be associated with bones, ligaments and joints rather than muscle tissues.[2] However, it is well known that muscular disuse causes muscular atrophy and a reduction in bone diameter. Since both of these phenomena occur with aging, it is difficult to determine whether slowness and decreased efficiency are due to the aging process or to disuse. It is probably a combination of both, in that activity levels decrease with age while the aging process makes it more difficult to maintain a sufficient level of physical activity.

From a structural standpoint, the older individual experiences a greater number of long-bone fractures, greater vertebral atrophy, and a greater number of compression fractures of the bone. It has been observed that the bone mass decreased with age in women but this has been not been brought out in men.[3] The density of the bone also appears to decrease with age in all races and both sexes.[4] This decrease in both mass and density of bone tends to explain the greater susceptibility of bone to fractures and dysfunction among the aged. Further research indicates that an increased reabsorption of calcium from bone into the blood stream occurs with age.[5] The reabsorption appears most noticeable in those bones that do not experience tension and stress. It has also been noted that a reduced ash content occurs as density decreases and the bones so affected are much more susceptible to injury.

The most common type of structural illness today is rheumatoid arthritis which is typically a problem of older age groups. It has been estimated that forty million people in the United States are afflicted with the disease, which is an inflammation of the joint and deterioration of the cartilage and bone in these locations. In many cases a very serious decrease in function occurs, which in turn decreases the level of activity. The joints are simply not capable of handling the tension and stress of physical activity without considerable pain and discomfort. The cause of the affliction is unknown at this time and the treatment is very often ineffective. The lack of function elicits disuse and disuse causes continued atrophy of the associated muscle tissue.

Motor efficiency and functional changes occurring during the aging process are very complex. On the one hand, the nervous pathways and receptors could cause this decrease in functionality and, on the other hand, it could be the reduction of the integrity of muscle tissue itself. The solution to the problem is hindered by the intervention of motivational factors in the measurement process. With older individuals it is very possible that the data generated are measuring the motivation of the subject to accomplish the task and not the motor efficiency or muscular function at all. This problem will have to be solved before current researchers will be able to deal effectively with interventions designed to improve efficiency and functon from a biological standpoint. It is generally agreed that training

programs using physical exercise will improve both of these conditions but we still do not understand why this is so.

A considerable amount of work has been done relative to muscular tissue change during the aging process. It has been observed that muscle weight decreases as a fraction of total body weight with age.[6] An increase in extra cellular water has been found to occur with an increase in sodium and chloride ion concentrations.[7] However, potassium concentrations have been found to decrease in the extracellular water space[8] while collagen and fat concentrations associated with muscle tissue increase with age. All of these factors most certainly affect both muscular efficiency and function.

The decrease in mass of muscle tissue appears to be due to a decrease in the number of fiber[9] as well as the diameter of these fibers.[10] Muscle fibers, or muscle cells, do not undergo cell division. Once a fiber is destroyed it is not replaced. The change in diameter would mean that protein substances are relased from the fiber, leaving less contractile (protein) substance available for muscular function. It is important to note that changes are not uniform in muscles of different functions. Types of motor units, given muscle groups, and individual muscle fibers vary a great deal in response to the aging process. Therefore, it appears that opportunity exists for the retardation of cell deterioration through intervention programs. As an example, the physically active diaphragm deteriorates very little as compared to a 39 percent decrease in the fibers of

a soleus muscle that is restricted in activity.[11] White muscle fibers decrease in both number and volume with age. The decrease in volume can be attributed to a decrease in the number of myofibrils within the fiber. The red muscle fibers appear to decrease in number only, leaving the assumption that the number of myofibrils in the fiber remains the same. The observation that white fibers may not decrease in number as rapidly as red fibers, could certainly create a condition seriously affecting the endurance of the aged tissue. Normal fibers both red and white tend to be very consistent in size in the young adult. A much greater variation in size occurs in fibers found within the aged muscle tissue. Another interesting fact is that the number of muscle spindles does not change with the decrease in size of given fibers. The number of muscle spindles present in muscle fibers of different parts of the body varies greatly but this number appears to stay the same regardless of the change in fiber diameter with age. The effectiveness of these spindles to provide sensory feedback as the aging process continues has not been studies.

The strength of a muscle group depends on the size and number of fibers within that muscle group. As we increase the size and number of fibers we increase strength and as we decrease size and number we reduce strength. Therefore, it could be the aging process that causes this change or it could be the lack of muscle activity in later years. That would also reduce the size and strength of muscle tissue.

Another factor that may be important to muscular strength is the decrease in anabolic hormone concentrations. These concentrations decrease with age and could cause a decrease in protein retention within the fibers. If we increase the concentration of hormone, we could then increase protein synthesis which would elicit a greater muscle size and strength. This procedure has been used successfully in drug therapy for older individuals who have been confined for a long period of time and unable to do normal daily chores. The administration of these hormones tends to elicit greater strength but only when the muscle tissue is challenged with an activity overload.

The change in physical function of the older individual is usually caused by both a decrease in strength and endurance. Most of this discussion thus far has related to the strength factor with little thought given to the endurance factor. In normal muscle tissue, the endurance of a muscle is related to the ability of the cardiovascular system to deliver oxygen which allows for adequate energy production. However, the oxygen supply does not appear to be a limiting factor for the aged performer, nor does the decreased mobilization of glucose to produce energy. However, this is not the case in cardiac and brain function, if the muscle tissue is sufficiently developed to provide a given level of function, it appears that the endurance factor will not be limiting to that function. This is somewhat of a surprise in that we recognize a very definite decline in the function

of the cardiovascular system in its ability to pump and distribute blood.

Although the skeletomuscular system is susceptible to the aging process after a plateau between the ages of 25 and 30, it is well documented that the functionality of individuals can be considerably above or below the expected capacity. An aerobic training program which involves muscular overload will increase oxygen uptake by muscle tissue, and increase the cellular activity which allows for both muscular strength and energy production. These changes will increase both strength and endurance of almost any muscle group regardless of the efficiency and functionality prior to the intervention program. The effect of training decreases with age but does not seem to be limited to a given age or functionality. As one grows older, it is harder to improve strength and endurance, particularly if the individual is in good functional condition prior to the intervention. It is unfortunate that so many older individuals suffer from hypokinetic (poor physical condition) disease when it can be avoided without serious risk or high incidence of injury.[12] This is not only true of the human but also in the animal kingdom where the aging process has been observed as considerably slower among wild animals as compared to domesticated animals living in a more sedentary environment.[13] Very definite differences have been noted in the effectiveness and viability of muscle tissues which in turn determines the effectiveness and viability of physical function.

Less is known about the aging of the neurosensory system than most other biological systems within the human body. Much of the research is conflicting and provides little basis for the true understanding of what occurs, much less for the cause of such occurrences. However, it is well known that nerve cells decrease in number after the age of twenty-five. There is a decreased capacity to send impulses from the brain but this decreased capacity is not uniform to all parts of the body and from all areas of the brain. In the case of the skeletomuscular system, those areas that are most active appear to age less rapidly. Conduction velocity also decreases with age and varies considerably among individuals of a given age and functionality.[14] The decrease in the effectiveness of the neurosensory system appears to be more of a concern for the control and function of the skeletomuscular system than it does for recall and reasoning processes that are specific to the neurosensory system itself.[15]

Along with the reduced motor control, it has also been found that sensory input to the central nervous system is impaired with age. It is entirely possible that these two are related because of the importance of sensory input to motor control. Some evidence would indicate that the problem occurs at the synapse where a greater concentration of inhibitor substance is produced as opposed to a decreased concentration of transmitting substance. Some researchers also have noted a decrease in the mylination of the peri-

pheral nerves. There appears to be a definite increase in lipofusin (age pigment) which is well known to be a factor in the aging process. However, it is not known whether the increase in lipofusin concentration is a result of the aging process or one of the causes of it. Efforts to reduce the concentration of this substance in tissue has not been effective in the reduction of the aging process.

Another factor relating to motor control is the change in sensory feedback from muscle spindles.[16] It has been noted that the number of intrafusal fibers in the spindle has decreased faster than the decrease in extrafusal fibers surrounding the spindle. If this is true, it would lead one to believe that the spindle would not be as effective in resetting itself in a new position for feedback after the muscle has begun to contract or relax. Finite motor movement would be less possible under these circumstances. This could be a possible reason for the slowness and less efficient motor movement patterns observed in the elderly.

Other factors associated with sensory input related to vision, hearing, and touch. Studies involving vision have indicated the existence of a less flexible lens which tends to yellow with time. This causes distortion in the image on the retina and also less color discrimination. The loss of sensitivity to high frequencies appears to be the first change occurring in audio sensitivity. The range of frequencies begins to diminish along with conductivity of auditory bone structures. It has also been noted that a

loss of hair cells in the corti occurs which would effect the sensitivity and transmission of the auditory response to the brain. Not as much is known about the propriocerebellar function which provides information about tissue stretch and the position of body segments, such as the lower arm. Decreases in nerve conduction velocity would make a difference in coordination but there is speculation that a decrease in the number of receptors or the effectiveness of these receptors also occurs. Most of the factors associated with sensory input to the brain could be related to a decreased blood supply in the area of the nerve cells or receptors. This is particularly true in the case of vision and hearing.

The connective tissues are made up of large molecules and intracellular space which form a very well-organized framework of collagen, elastin, and several other large molecules. The interaction of these molecular structures varies with function. Research has been difficult because of the inability to get tissue samples over a period of time. We have only scratched the surface in our quest to understand the change and function of connective tissues as they relate to the aging process. Only collagen and elastin have been studies to the extent that generalizations can be made at this point in time.

The physical properties of connective tissue change greatly with time.[17] A great deal of change occurs in the first five years of life with moderate change occurring

between the age of ten and thirty. After the age of forty, a slow degenerative change occurs which decreases the diameter of the tissue cells.[18] The decrease in the volume of cartilage begins between the ages of twenty and thirty in both weight-bearing and non-weight bearing body segments. The color of the tissue changes from translucent bluish to opaque yellow. Changes on the surface of cartilage include cracking, fraying, and shredding. In addition, progressive cell death occurs[19] and irregular bodies are formed causing a mild dysfunction which continues to become more critical. The depth and diameter of tissue continues to decrease and the percent of water decreases about 40 percent with advanced age.[20]

Collagen is one of the most abundant structures in connective tissues, which tends to hold it together. It becomes much more stable, less eleastic with age, and therefore less responsive to mechanical stress. This may be due to chemical activity in the protein which results in the cross linking of large molecules.[21] This cross linking tends to bind the protein strands together and the greater the maturation the more stable the crosslinks tend to be. This process, which seriously limits tissue flexibility, has been related to diet.[22] Studies have also indicated that physical activity levels affect the condition of this tissue. Among young individuals activity normally experienced will provide for periodic muscle overload, increase the strength, and maintain the viability of the connective tissues. This overload is often not present in the aged.

Elastin also undergoes a predictable chemical modification with age. Chemical crosslinks appear to form similar to those of collagen.[23] This causes a similar decrease in flexibility of the tissues, and therefore, a reduced range of motion at the joint as well as greater susceptibility to injury.

One of the most abundant connective tissues is the skin. The tension of the skin increases with maturation and reaches an all-time high at puberty. Then a slight decrease in this tension occurs which is followed by a slow increase, with age, after that point in time. An increase in collagen concentraton in skin occurs which decreases the elasticity and flexibility of the tissue. It has also been noted that a decrease in microcirculation occurs along with a decrease in fat content. It appears that more of the tissues of the skin are replaced with collagen during the aging process and the collagen then becomes cross-linked,[24] causing reduced flexibility.

An increase in the incidence in skin infection is related to the decrease in immune system activity of the aged. A concomitant loss of sweat gland and lubrication gland activity occurs which increases the rididity of the tissue. The tissues of heart valves react in a similar way to the aging process with the increased collagen concentration and a decreased number of cells. Little information is available on function as it relates to this change but it is well known that the pumping action of the heart decreases with age.

The aging process of connective tissues is interesting because cells produce the large molecules found in intracellular space that form the organized framework of this tissue. It is important to determine if it is the cell that produces poor quality molecular substances with age or if the cell produces the same type of molecular substance with age that it does earlier in life and the structural matrix then changes as a functioning substance. At the present time, we have little information that would help us resolve this mystery. It is, however, important to realize that connective tissue responds to its environment. Both volume and strength will change as the conditions change. This provides an excellent opportunity for prevention since we can change the environment externally to create the desired response. This response may be counter to the aging process and therefore retard the rate of change.

The failure of the cardiovascular system to function effectively accounts for more than one-half of the deaths in our society today. The aging process that occurs in the cardiovascular system is progressive and physiologically irreversible. The components of this aging process are varied, but fortunately understood to a greater degree than most other physiologic systems.

As in other tissues, the concentration of lipofusion or, "age pigment" increases in the tissues of the heart.[25] This increase occurs at a rate of .3% to .6% per decade of life. The effect of this concentration continues to be a

mystery but the changes in concentration are well documented. The heart also experiences a replacement of muscle tissue with connective tissue.[26] The rate at which this occurs has not been quantitatively established but the loss of this muscle tissue has been considered responsible for loss of forceful contraction of the muscle tissue in the heart. The loss of cells has not been noted in the decrease of muscle tissue in the heart with age. Some hearts actually get bigger and some get smaller. This is quite predictable in that the hearts that lose muscle tissue tend to decrease in size but when the connective tissue becomes fatigued and possible hypertension occurs within the system, the heart begins to increase in size due to the greater pressure. This causes a decrease in the force of contraction and eventually the pumping action of the heart. It has also been noted that a loss of nervous tissue occurs in the heart. The nodale system that controls the electrical activity of the heart, and therefore, the rate of contraction, decreases with a concomitant increase in collagen. The decrease in nervous tissue appears to occur with the loss of cells in the nodale areas.

A reduction in cardiac output of the heart with age has also been observed. This change occurs at the rate of approximately one percent per year after twenty years of age. The heart rate decreases per unit time and the stroke volume also decreases.[26] This condition eventually reduces the profusion of tissues within the body of blood and there-

fore reduces the supply of oxygen for energy production in cells. Among those tissues affected is the heart muscle itself. This decreases the ability of the heart to produce energy and, therefore, to maintain effective contractility. This problem is most often complicated with a decrease in the size of the coronary arteries due to arterial problems.

The end result of decreased cardiac output is a decrease in the ability of the heart to function as a pump. The force of contraction is reduced along with the velocity of blood flow creating a less responsive flow of blood to critical tissues.[27] The heart also becomes less responsive to neurohumors in the blood which under normal conditions will increase the force of contraction and thereby the cardiac output. However, this lack of responsiveness to hormonal agents means that the heart does not speed up under physical and emotional stress as much as it would normally and therefore it may conserve badly needed oxygen and energy reserves. The system would experience fatigue at a faster rate under these sets of circumstances but would also conserve needed supplies of oxygen and energy and possibly reduce the risk of a heart attack.

It is very encouraging to note that the changes that have been described thus far do not occur as intensely in people who have achieved a reasonable or high degree of physical fitness.[28] Individuals who have maintained a relatively active life have continually challenged the heart in such a way that the volume of tissue reduction is mini-

mized. It is also encouraging to note that thoe individuals who are recovering from a cardiovascular indicent may improve cardiovascular function through exercise rehabilitation programs.[29] This type of program may not be appropriate for all cardiovascular patients and certainly should be carried out within the confines of a well-supervised and monitored situation. A very complete evaluation system of cardiac condition and pathology is also necessary. Several studies have indicated that the physiologic change which occurs with such a rehabilitation program using increased physical activity and specifically indicate the physiologic changes that occur as a result of these prevention programs. These changes tend to improve the function of the heart as a pump and reduce the effect of the aging process. Most epidemiological studies indicate that there is two or three times the risk of a cardiovascular problem among sedentary individuals as opposed to physically active individuals. These studies have been conducted using subjects of all ages including those in their sixties and seventies.

The condition of the systemic arterial system is also critical to cardiovascular function. It has already been noted that increased resistance in the arteries has an effect on the pressure within the heart. This increase in resistance is due largely to more rigid and smaller arterials which increases the pressure within the system.[30] The replacement of smooth muscle by connective tissue in the arteriols causes a loss of elasticity and an increase in

rigidity. This condition is called arteriosclerosis and is one of the critical factors associated with high blood pressure. Elevated blood pressure indicates hypertension which is observed in most aged individuals and considered one of the major health problems associated with age.[31,32]

Another condition of the arteries, atherosclerosis, is due to a plaquing process which occurs within the layers of the arterial wall. As lipid (fat) substances move through the arterial wall they sometimes become lodged within the tissues and begin to create a softening condition within these tissues. Later this softening condition becomes hard and a plaque is formed which reduces the size of the interior space of the vessel. This reduces the blood supply and also the ability to transport oxygen to tissue farther from the heart.[33] When the heart is less effective as a pump, and less efficent, it needs more oxygen to do the same amount of work that it did at a younger age. At the same time, the blood, and therefore oxygen, supply to the heart muscle is decreased. When the energy supply is less than the energy needed to keep the heart going, the "usual" kind of heart attack occurs, which is called a myocardial infarction. Although this condition represents our greatest health hazard today for older individuals, the projection of future problems is even greater. The plaquing process is presently irreversible and most elderly individuals of today were not observed to have this plaquing conditon as young adults. Now, this condition is observed in most of our young adults as well.

An added problem is that advancing age produces a decreased response to pressure changes within the arterial system. As the arteries become less flexible and the pressure increases, the body does not respond normally to reduce the pressure. In most cases, the pressure continues to increase as the aging process continues.

Rather encouraging results have been observed when using physical exercise as a means of reducing the incidence of arterial rigidity and plaquing. The evidence is not as complete and conslusive as that of the heart response to exercise but most researchers would agree that very positive changes can occur with regular and appropriate physical activity. Although the condition is not reversible, it has been noted that the condition can be retarded through physical exercise and certain types of dietary intervention.

The function of the immune system is to protect the body and maintain the integrity of the tissues that make up the body. The tissues involved are the thymus, the spleen, and the lymph nodes. The immune system prevents the loss of tissue integrity from the body's own production of foreign materials as well as those materials that enter from outside the body. The immune system is made up largely of the reticular endothelial structures which consist of two kinds of cells, the T cells and the A & B cells.[34] The T cells go through the thymus prior to biological activity. The thymus is located above the heart and is quite susceptible to steroids and stress which will reduce its size and

function. The A & B cells do not go through the thymus but act directly within the blood stream.

If a foreign bacteria (antigen) enters the body through the intestinal area, A cells (white cells) engulf the bacteria and digest it. The waste is encapsulized in the white cell which goes back to the lymph node. At the node, the white cell stimulates B cell production which is specific to the given foreign substance and the B cell goes out into the blood to destroy more antigens. The A cell also leaves memory cells in the lymph node for a secondary defense. This is the reason that immunizations are very important. The A cells pick up the bacteria and take it to the lymph node where specific B cells are formed that are ready and waiting to attack the specific invader when and if it should appear. The T cells act as helpers or suppressors of the defense process,[35] and the A & B cells are largely responsible for the actual defense against foreign bodies.

It has been well documented that the immune system is not as effective in aged populations.[36] Young adults have little problem in producing new A & B cells and can therefore ward off most invading substances. However, with age the ability to produce new cells that attack bacteria and other foreign substances is reduced to approximately 50% of those produced at a younger age. The problem is magnified by the fact that the A & B cells that are produced are not as effective in function, as well as being fewer in number. This degradation process occurs shortly after sexual matur-

ity when the thymus gets smaller and less active. As a result, the substances secreted by T cells that help or suppress A & B cell activity do not enhance the fight against foreign bodies.[37] The suppressants appear to be more prevalent in older age, hindering the activity of the A & B cells to respond quickly.[38] It does not appear that these cells wear out but that the activity and regeneration process is suppressed. The organisms are then much more susceptible to invasion by bacteria and much less able to cope with foreign bodies that previously were quickly neutralized.

It was mentioned earlier that the immune system may also attack substances produced by the body itself. Autoimmunity means that the immune system indiscriminately attacks and destroys the body's own molecular substances to the point that it is difficult for the tissues to survive. The suppression that normally comes from the T cells appears to be lost and the aggressive A & B cells attack substances throughout the body that are necessary for normal functioning. The older person is more susceptible to this danger than the younger individual.

It has been said that there are as many theories associated with the biological aging process as there are researchers studying the phenomenon. Although the literature would support this position to some extent, a careful review of the theories provide an opportunity to classify them into groups which share citical concepts and observations. Most of these theories will fit within two basic categories which

will be described as unprogrammed theories and programmed theories. Those theories that are unprogrammmed subscribe to a random series of events that are unplanned but contribute to the aging process as time passes. The programmed theories lean toward genetic factors and events that are programmed prior to birth and implemented through central nervous system and the endocrine system.

One of the most popular unprogrammed theories of the aging process is that of somatic mutation. This theory takes the position that the aging process is an accumulation of defects occurring within the gene (chromosomes) that result in a loss of information at a particular spot on the molecule. Radiation is one of the greatest causes of this problem. If the cell divides, as some tissues do, the daughter cells will not be the same as the parent cell and therefore, will not function effectively in its predetermined role. If a cell does not divide but continues to turn over its intercellular substances, the loss of the message on the chromosome will tend to bring in ineffective materials which also causes the production of poor quality cells and a loss of cellular function. The more poor quality cells or materials within cells, the greater the dysfunction of the resulting organism.

This theory is supported by the position that it is also possible for chemical substances, called free radicals, to be present within the cell that will chemically react with the chromosome to cause the same problem as described

above. Many chemical substances have been administered to bind up the free radicals which tend to cause this illicit chemical activity. However, this process has been found to be ineffective and free radicals remain ready and willing to react with chromosomes resulting in a continuation of the aging process. Some gerontologists feel that this chemical activity occurs through a crosslinking of the chromosomes substances and not through free radicals at all. This too may cause an inactivation of the genetic material that produces new substances within the cell and thereby result in a reduction of functionality.

The error-catastrophy theory is similar to the somatic-mutation theory in that it also addresses itself to the alteration in the molecular biology of the cell as the materials within the cell turn over and new substances are formed to continue the cellular activity. The message indicating what kind of materials to bring into the cell comes from the chromosome which is located in the nucleus of the cell. The genetic material sends out another substance which carries this message to the other parts of the cell and is responsible for bringing in the right kind of protein substances to produce the appropriate type of protein within the cell. The error-catastrophe theory states that the message carrying the substance becomes contaminated, or the message becomes confused, and the wrong proteins are brought into the cell thus producing materials that are ineffective in conducting the cellular activities. After maturity, this

process continues to occur until a large number of cells have become incapacitated and therefore hinder the functionality of the total individual.

The protein-crosslinking theory is similar to the others but has some very distinct differences. This theory takes the position that collagen crosslinks with the protein of the cell and this process continues with age. The original cellular protein production occurs uninterrupted but once the protein is formed, the chemical crosslinking occurs with collagen in such a way that the molecule becomes stronger but less flexible. If this happens in a muscle cell, the contractibility of the cell is impaired considerably. It also affects the chemical activity of the protein substance within the cell. The elasticity of the tissue, particularly in tendons and arteries decreases considerably, even to the point of replacing viable muscle tissue with scar tissue. An example of this loss of flexibility can be seen in the skin-pinch test. In the young individual this pinch quickly diappears but in the older individual the effect of the pinch remains because the flexibility of the tissue has been decreased. For many years, scientists have been trying to find an enzyme that will break the crosslinking bonds that tend to increase with age and decrease human functionality. All efforts to do this have been unsuccessful to this point in time.

Another theory that is quite different from these, yet unprogrammed in nature, is the auto-immunity theory. This

theory states that the antibodies making up the immune system within the body attack the organism's own protein in addition to foreign protein substances that might enter the body from different sources. In other words, the immune system racts against its own tissue and, in a sense, rejects itself. This activity is considered random and increases with age. However, it must be realized that this condition might also be programmed to the point that it is nature's way of completing the life cycle. This theory relates to the others in that antibodies are also proteins and, if the molecular substances within the proteins are malformed due to a poor quality messenger substance, the malformed antibodies do not function as they should and erroneously attack the tissues that are needed for total body function rather than the invading antigens.

One of the oldest theories of aging is the lipofusion-pigment theory. It has been known for years that lipofusion, also called "age pigment," increases with age. This occurs mainly in the heart and brain. The substance is made up chiefly of protein and lipid chemical substances but the specific chemistry is unknown. The function of the lipofusion pigment within the cell is also unknown but certain chemical substances tend to decrease the concentration. Some researchers think that an accumulation of the lipofusion pigment causes the aging process to occur. However, it must be noted that this substance could also be a result of the aging process since as aging continues the concentration increases. It

could also be an expression of the cell's failure to metabolize to form energy. Some researchers have also related this process to free radical activity because it appears to be related to intercellular oxidation which is part of the metabolic process.

Another, and very old, theory is that of calcium shift which has been postulated by those scientists who have related stress to the aging process. It is well known that the aging process occurs as a result of, or along with, a change in soft to harder and less flexible tissues. At the same time, bones get weaker and decrease in both density and mass. This circumstance makes it easy to speculate that calcium, which is largely responsible for healthy bone, moves from the bone tissues to soft body tissues and makes the bone weaker and the soft tissues more ridid. The aging process, then is here considered to be an accumulation of this shift over a period of time, resulting in decreased function of the soft tissues and more fragile bone structure. There is some evidence that this shift tends to occur at a greater rate as stress and body tension increases. However, no attempt has been made to support this theory with data from controlled experimentation.

The intervention of the aging process, if the process is unprogrammed, would come through the reduction of accidents that have detrimental effects on biological function. It is important that these accidents be more clearly defined and that we have greater data to support one or the other of

these theories before effective intervention can occur. It is interesting to note, however, that these theories have all been postulated since the Second World War, although civilizations have been concerned about the aging process for thousands of years. It is easy for even the non-researcher to see that one or two scientific breakthroughs in aging-process research could effect a rather significant change in the lonevity of human beings, as well as improve functioning of the physical organism during the first 70 or 80 years.

Now let's look at programmed theories of aging. Cell division is programmed to a very specific degree during the development states of the human life span. The mitotic-exhaustion theory states that this specificity continues through the aging process and that cells can only divide a certain number of times. When the cell gets close to the end of its capacity to divide, many things begin to happen that result in illnesses and incapacities. If the organism is able to survive these challenges, then the final stage of life is ended through the inability of the cells to divide and maintain life. This theory is related to all cells except nerve and muscle cells. It is strongly suggested that this process is pre-programmed and that it is as much a part of the life cycle as the developmental stage. The mechanism through which this occurs has not been described but it is thought to be triggered in some way when the reproductive phase of life has reached its plateau.

Another of the programmed theories of the aging process is the rate-of-living theory. According to this theory, there is a given amount of energy available to the intact human and just so many heartbeats available during a lifetime. We simply run out of energy and therefore the heart and vital organs stop functioning. Smaller organisms have a higher heart rate and a reduced lifespan. Larger animals, like the elephant, have a low heart rate and a very long lifespan. Little information is available to support this theory among humans which have either a very low resting heart rate or a very high resting heart rate. It is, however, interesting to note that those individuals who are in better physical condition have a lower resting heart rate and there is some indication that they have greater longevity. Although this theory appears to make sense among the total order of the animal kingdom, no attempt has been made to relate this theory to humans or to apply it to one group of humans as compared to another. Those persons that support the theory also make note of the fact that brain weight is, related to body size and that this relationship is a good measure of longevity. As the brain weight per unit of body weight increases, so does longevity within species of the animal kingdom. Therefore, the combination of lifetime energy, brain complexity, and body weight may be a factor in the longevity of the human.

The programmed theory that has the most support at present and appears to be gaining support among researchers

is the central-nervous-system-endocrine theory. This theory states that aging is just another stage of development and that the same process that turns on certain activities during development turns them off after sexual maturity. This means that there is a regulated control of the phases of life and that the aging process is not a series of accidents or unprogrammed incidents. This does not mean that we cannot do something about the aging process. We know a great deal about hormonal control and what triggers the onset of maturity, which means that we may also learn about those events that trigger maturation and aging. Once we know the process and the mechanism for control, we can then begin to alter it positively. It is understood that other factors are superimposed on this programmed aging process. Stress, nutrition, exercise, and other factors affect the rate and the mechanism associated with programmed aging.

We can learn a great deal about programmed aging from lower forms of animal life. In animal studies, we see the same possible hormonal activity controlling the rate of change from one developmental stage to another. This process of metamorphosis starts from the brain and affects changes within the genetic makeup of the cell. We see the insect evolve from the egg to the larvae stage, then to the pupas (cocoon stage), and finally to the adult insect stage. Specific genes are engaged at given times and agin occurs in a programmed way because of the cell environment that turns on a certain portion of the genertic substance. We may be

able to turn on and turn off this genetic message to the cell in such a way that the aging process can be altered to maintain integrity of tissues, and thereby affect lonevity and functionality. The fact that we do not understand these mechanisms completely at this time means that more research relating to this process will be necessary before we can develop intervention strategies.

Although some scholars still feel that the aging process is finite and predictable, it is obvious from our review of the theories of aging that we have learned a great deal in the last twenty years and are still not able to predict this process. If this explosion of knowledge continues, we can expect, in the not too distant future, additional breakthroughs and support for certain of the theories presented. Although we have a long we to go, we now understand the nature of the biological aging process much better than a few years ago. It appears reasonable that an even greater knowledge of the nature of the biological aging process could lead to an improvement in the human condition, and possibly to increased longevity. Regardless of how we feel about intervening in the aging process through hormonal control mechanisms, it is also possible to affect the aging process through more natural intervention tactics. Changing a person's lifestyle to include better nutritional and activity habits may be a more widely acceptable strategy for promoting better health. It appears probable that interventions such as these could improve longevity, but it is

almost certain that such practices can delay the effects of aging and improve the quality of life during the later years.

Interveing in the aging process has taken many forms through the years. Until only a few years ago, interventions were highly mystical and in many cases profit-motivated. More recently, aging and the biological theories that describe it have been carefully studies to determine possible intervention techniques that are scientifically based. In describing the nature and theoretical basis of the aging process, this paper has suggested some appropriate techniques.

Drug-oriented interventions have not been demonstrated to be an effective solution except in disease-oriented conditions. On the other hand, nutrition and exercise appear to be the most acceptable and effective interventions. If the nutrition of an individual is inappropriate, the effect of aging is magnified. A good diet will help the body resist disease and avoid abnormally rapid aging, but it will not necessarily improve the function of the various systems that have been discussed.

Physical activity, on the other hand, has been shown to improve one's physical condition and make it possible for the older person to live a more complete and satisfying life. The most important issue is the intensity and duration of physical activity, the objective being to maximize development and minimize injury. This is why an older

person must consult a physician prior to changing his or her level of activity. The physician will be able to diagnose the existence of possible pathology that may be affected by this change in lifestyle. Before changing one's level of activity, one should have a work-capacity evaluation and receive a prescription for an exercise program. If this is not done, the person may undertake a level of activity that is not sufficient to elicit the type of change that will improve physical condition. Conversely, the person may undertake a too-aggressive exercise program that is actually harmful and possibly lethal.

Organized exercise programs conducted by trained professionals will help to eliminate the possibility of too little or too much exercise. These programs usually include warm-up, flexibility, relaxation, and aerobic activities that are designed to stimulate and improve the cardio-pulmonary system. Aerobic activities are total body activities such activities such as walking, running, cycling, and swimming. Not only do they add more oxygen to the tissue, but they also improve the efficiency of oxygen utilization. Greater oxygen supply provides more energy production, and increased energy production reduces fatique. This is particularly important to the heart which needs a great deal of energy to understand both physical and emotional stress. In addition to cardio-pulmonary conditioning, appropriate physical exercise can also enhance an older person's strength, hand-eye coordination, reaction time, and muscle tone.

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